08/918 40 F
Att #9

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**********
FILE 'USPAT' ENTERED AT 12:01:16 ON 10 MAR 1999

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*  WELCOME TO THE  *
*  U.S. PATENT TEXT FILE  *
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=> s p53

L1  1025 P53

=> s adenovir?

L2  4162 ADENOVIR?

=> s l1(5w)l2

L3  19 L1(5W)L2

=> s l1(5a)l2

L4  26 L1(5A)L2

=> d l4,cit,rel,ab,1-26
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1. 5,877,011, Mar. 2, 1999, Chimeric adenoviral vectors; Donna Armentano, et al., 435/320.1; 536/23.72 [IMAGE AVAILABLE]

US PAT NO: 5,877,011 [IMAGE AVAILABLE] L4: 1 of 26

ABSTRACT:
A chimeric adenoviral vector is provided that comprises nucleotide sequence of a first adenovirus, wherein at least one gene of said first adenovirus encoding a protein that facilitates binding of said vector to a target mammalian cell, or internalization thereof within said cell, is replaced by the corresponding gene from a second adenovirus belonging to subgroup D, said vector further comprising a transgene operably linked to a eucaryotic promoter to allow for expression therefrom in a mammalian cell. Additionally, a method of delivering transgenes to target mammalian cells, particularly airway epithelial cells, is provided.

2. 5,876,711, Mar. 2, 1999, Methods and compositions for determining the tumor suppressor status of cells; Ali Fattaey, 424/93.2; 435/5, 6; 514/44 [IMAGE AVAILABLE]

US PAT NO: 5,876,711 [IMAGE AVAILABLE] L4: 2 of 26

ABSTRACT:
Methods and compositions for determining the tumor suppressor status of cells are described, preferably as pertaining to the p53 status of tumor cells, and preferably in vivo using a recombinant construct consisting of a first polynucleotide sequence that encodes a reporter molecule and a second p53 binding polynucleotide sequence that is operably linked to the first polynucleotide sequence such that binding of p53 to the second polynucleotide sequence causes the expression of the reporter molecule which can be detected or quantified.

3. 5,874,235, Feb. 23, 1999, Screening assays for cancer chemopreventative agents; Timothy A. Chan, et al., 435/18, 4, 7.21, 7.23, 19, 21; 436/63 [IMAGE AVAILABLE]

US PAT NO: 5,874,235 [IMAGE AVAILABLE] L4: 3 of 26

ABSTRACT:
Nonsteroidal anti-inflammatory drugs cause a dramatic increase in intracellular ceramide, which induces apoptosis. The ceramide increase is likely mediated by cyclooxygenase inhibition, which elevates arachidonic acid, which stimulates sphingomyelinase, which produces ceramide. Contacting members of this pathway with test compounds and observing their effects provides a method of screening for potential cancer chemopreventative agents.

4. 5,863,795, Jan. 26, 1999, Nucleic acids that encode peptides which modulate apoptosis; Thomas D. Chittenden, et al., 435/325, 243, 320.1, 410; 536/23.5, 24.31 [IMAGE AVAILABLE]

US PAT NO: 5,863,795 [IMAGE AVAILABLE] L4: 4 of 26
REL-US-DATA: Division of Ser. No. 440,391, May 12, 1995, Pat. No. 5,656,725.

ABSTRACT:
The present invention is directed to novel peptides and compositions capable of modulating apoptosis in cells, and to methods of modulating apoptosis employing the novel peptides and compositions of the invention. In one aspect, the invention is directed to a novel peptide designated the "GD domain," which is essential both to Bak's interaction with Bcl-x.sub.L, and to Bak's cell killing function. Methods of identifying agonists or antagonists of GD domain function are provided. The GD domain is responsible for mediating key protein/protein interactions of significance to the actions of multiple cell death regulatory molecules.

5. 5,858,987, Jan. 12, 1999, E6AP antisense constructs and methods of use; Peggy L. Beer-Romero, et al., 514/44; 435/5, 6, 91.2; 536/23.1, 24.3, 24.33, 24.5 [IMAGE AVAILABLE]

US PAT NO: 5,858,987 [IMAGE AVAILABLE] L4: 5 of 26

ABSTRACT:
The present invention relates to the discovery that antisense nucleic acids complimentary to an E6AP gene can be used to regulate cellular p53 levels. In general the invention features E6AP antisense constructs which, by inhibiting E6AP activity, can modulate cellular p53 levels in both p53+ transformed cells and in normal cells. The invention also provides methods for treating papillomavirus (PV) induced condition, methods for regulating cellular p53 levels and methods for regulating cellular proliferation.

6. 5,856,181, Jan. 5, 1999, Cytopathic viruses for therapy and prophylaxis of neoplasia; Francis McCormick, 435/325, 235.1, 375; 536/23.1, 24.1 [IMAGE AVAILABLE]

US PAT NO: 5,856,181 [IMAGE AVAILABLE] L4: 6 of 26
REL-US-DATA: Continuation of Ser. No. 641,081, Apr. 29, 1996, Pat. No. 5,677,178, which is a continuation of Ser. No. 198,184, Feb. 16, 1994, abandoned, which is a continuation-in-part of Ser. No. 17,525, Feb. 16, 1993, abandoned.

ABSTRACT:
Methods and compositions for treating neoplastic conditions by viral-based therapy are provided. Mutant virus lacking viral proteins which bind and/or inactivate p53 or RB are administered to a patient having a neoplasm which comprises cells lacking p53 and/or RB function. The mutant virus is able to substantially produce a replication phenotype in neoplastic cells but is substantially unable to produce a replication phenotype in non-replicating, non-neoplastic cells having essentially normal p53 and/or RB function. The preferential generation of replication phenotype in neoplastic cells results in a preferential killing of the neoplastic cells, either directly or by expression of a cytotoxic gene in cells expressing a viral replication phenotype.

7. 5,846,945, Dec. 8, 1998, Cytopathic viruses for therapy and prophylaxis of neoplasia; Francis McCormick, 514/44; 435/235.1, 320.1, 325, 375; 536/23.1, 24.1 [IMAGE AVAILABLE]

US PAT NO: 5,846,945 [IMAGE AVAILABLE] L4: 7 of 26
REL-US-DATA: Continuation of Ser. No. 198,184, Feb. 16, 1994, abandoned, which is a continuation-in-part of Ser. No. 17,525, Feb. 16, 1993, abandoned.

ABSTRACT:
Methods and compositions for treating neoplastic conditions by viral-based therapy are provided. Mutant virus lacking viral proteins which bind and/or inactivate p53 or RB are administered to a patient having a neoplasm which comprises cells lacking p53 and/or RB function. The mutant virus is able to substantially produce a replication phenotype in neoplastic cells but is substantially unable to produce a replication phenotype in non-replicating, non-neoplastic cells having essentially normal p53 and/or RB function. The preferential generation of replication phenotype in neoplastic cells results in a preferential killing of the neoplastic cells, either directly or by expression of a cytotoxic gene in cells expressing a viral replication phenotype.

8. 5,843,737, Dec. 1, 1998, Cancer associated gene protein expressed therefrom and uses thereof; Lan Bo Chen, et al., 435/455, 6, 183, 320.1,

325, 477; 536/23.1, 23.2, 23.5 [IMAGE AVAILABLE]

US PAT NO: 5,843,737 [IMAGE AVAILABLE] L4: 8 of 26

ABSTRACT:
We have now discovered that eukaryotes, including mammals, have a gene that encodes a multifunctional protein having helicase activity, DNA repair activity, p53 sequestering activity and oncogenetic transformation potential. Enhanced transcripts and expression of this gene in non-testicular cells have a high correlation to disease state in a number of cancers, such as colorectal carcinomas, hereditary cancers resulting from defects in DNA repair pathways, breast cancers, etc. Accordingly, discovering enhanced levels of transcript or gene product in non-testicular tissues can be diagnostic of a predisposition to cancer, and prognostic for a particular cancer.

9. 5,843,659, Dec. 1, 1998, Apoptosis gene EI24, compositions, and methods of use; Sophie M. Lehar, et al., 435/6, 69.1, 91.4, 320.1, 325; 536/23.5 [IMAGE AVAILABLE]

US PAT NO: 5,843,659 [IMAGE AVAILABLE] L4: 9 of 26

ABSTRACT:
Disclosed is the isolation and characterization of EI24, a novel gene whose 2.4 kb mRNA is induced following etoposide treatment. Induction of EI24 mRNA by etoposide required expression of wild-type p53. Overexpression of functional p53 was sufficient to induce expression of the EI24 mRNA. The EI24 mRNA was also induced in a p53-dependent manner
by ionizing irradiation of primary murine thymocytes. The invention is thus directed to an isolated EI24 protein, nucleotide sequences coding for and regulating expression of the protein, antibodies directed against the protein, and recombinant vectors and host cells containing the genetic sequences coding for and regulating the expression of the protein sequence. The invention is also directed to genomic DNA, cDNA, and RNA encoding the EI24 protein sequence and to corresponding antisense RNA sequences. Antibodies can be used to detect EI24 in biological specimens, including, for example, human tissue samples. The present invention is further directed to methods of treating degenerative disorders characterized in inappropriate cell proliferation or inappropriate cell death. The present invention is further directed to methods for diagnosing degenerative disorders characterized in inappropriate cell proliferation or inappropriate cell death, as well as methods for monitoring the progress of such degenerative disorders.

10. 5,837,520, Nov. 17, 1998, Method of purification of viral vectors; Paul W. Shabram, et al., 435/239, 69.1, 235.1, 320.1, 803 [IMAGE AVAILABLE]

US PAT NO: 5,837,520 [IMAGE AVAILABLE] L4: 10 of 26

ABSTRACT:
The invention provides a method for purifying viral vectors containing therapeutic genes for use in gene therapy. The invention comprises a method of purification from a cell lysate of a recombinant viral vector containing a therapeutic gene, which comprises: a) treating said lysate with an enzymatic agent that selectively degrades both unencapsulated DNA and RNA; b) chromatographing the treated lysate from step a) on a first resin; and c) chromatographing the eluant from step b) on a second resin; wherein one resin is an anion exchange resin and the other is an immobilized metal ion chromatography (IMAC) resin.

11. 5,824,544, Oct. 20, 1998, Adenovirus vectors for gene therapy; Donna Armentano, et al., 435/320.1; 514/44 [IMAGE AVAILABLE]

US PAT NO: 5,824,544 [IMAGE AVAILABLE] L4: 11 of 26
REL-US-DATA: Continuation-in-part of Ser. No. 409,874, Mar. 24, 1995.

ABSTRACT:
The present invention relates to novel adenovirus vectors for use in gene therapy which are designed to prevent the generation of replication-competent adenovirus (RCA) during in vitro propagation and clinical use. The invention also provides methods for the production of the novel virus vectors. These vectors maximize safety for clinical applications in which adenovirus vectors are used to transfer genes into recipient cells for gene therapy.

12. 5,820,868, Oct. 13, 1998, Recombinant protein production in bovine adenovirus expression vector system; Suresh K. Mittal, et al., 424/199.1, 233.1; 435/235.1, 320.1 [IMAGE AVAILABLE]

US PAT NO: 5,820,868 [IMAGE AVAILABLE] L4: 12 of 26

ABSTRACT:
The present invention relates novel live bovine adenovirus (BAV) expression vector systems in which part or all of one or both of the early region 1 (E1) and early region 3 (E3) genes are deleted and replaced by a foreign gene or fragment thereof and novel recombinant mammalian cell lines stably transformed with BAV E1 sequences, and therefore, express E1 gene products capable of allowing replication therein of a bovine adenovirus having an E1 deletion replaced by a heterologous nucleotide sequence encoding a foreign gene or fragment thereof and their use in production of (antigenic) polypeptides or fragments thereof for the purpose of live recombinant virus or subunit vaccine or for other therapies.

13. 5,814,452, Sep. 29, 1998, Human prostatic cell lines immortalized by adenovirus 12-simian virus 40 (AD12/SV40) hybrid virus; Mukta M. Webber, et al., 435/6, 371 [IMAGE AVAILABLE]

US PAT NO: 5,814,452 [IMAGE AVAILABLE] L4: 13 of 26
REL-US-DATA: Division of Ser. No. 234,981, Apr. 28, 1994, Pat. No. 5,610,043.

ABSTRACT:
Immortalized non-tumorigenic or tumorigenic human prostatic epithelial and fibroblast cell lines and derivatives thereof, containing DNA of a hybrid virus between adenovirus 12 and simian virus 40. The cell lines are useful for research on causes, treatment and prevention of prostate cancer, benign prostatic hyperplasia, male infertility, birth defects, aging and assessment of environmental toxic agents.

14. 5,811,281, Sep. 22, 1998, Immortalized intestinal epithelial cell lines; Andrea Quaroni, et al., 435/353, 320.1, 467 [IMAGE AVAILABLE]

US PAT NO: 5,811,281 [IMAGE AVAILABLE] L4: 14 of 26
REL-US-DATA: Continuation-in-part of Ser. No. 89,847, Jul. 12, 1993, abandoned.

ABSTRACT:
Novel intestinal epithelial cell lines having stably incorporated heterologous DNA having a temperature-sensitive mutant oncogene are described, wherein the cell line proliferates at permissive temperatures in a conditionally immortalizing phenotype; and ceases to proliferate at nonpermissive temperatures thereby effecting cessation of cell proliferation and a differentiated intestinal epithelial cell phenotype including expression of certain brush border enzymes, and keratin markers.

15. 5,801,029, Sep. 1, 1998, Cytopathic viruses for therapy and prophylaxis of neoplasia; Francis McCormick, 424/93.2, 93.3, 93.6; 435/235.1, 236, 320.1, 467; 514/44 [IMAGE AVAILABLE]

US PAT NO: 5,801,029 [IMAGE AVAILABLE] L4: 15 of 26
REL-US-DATA: Continuation of Ser. No. 198,184, Feb. 16, 1994, abandoned, which is a continuation-in-part of Ser. No. 17,525, Feb. 16, 1993, abandoned.

ABSTRACT:
Methods and compositions for treating neoplastic conditions by viral-based therapy are provided. Mutant virus lacking viral proteins which bind and/or inactivate p53 or RB are administered to a patient having a neoplasm which comprises cells lacking p53 and/or RB function. The mutant virus is able to substantially produce a replication phenotype in neoplastic cells but is substantially unable to produce a replication phenotype in non-replicating, non-neoplastic cells having essentially normal p53 and/or RB function. The preferential generation of replication phenotype in neoplastic cells results in a preferential killing of the neoplastic cells, either directly or by expression of a cytotoxic gene in cells expressing a viral replication phenotype.

16. 5,789,538, Aug. 4, 1998, Zinc finger proteins with high affinity new DNA binding specificities; Edward J. Rebar, et al., 530/324, 300, 350, 358, 400 [IMAGE AVAILABLE]

US PAT NO: 5,789,538 [IMAGE AVAILABLE] L4: 16 of 26
REL-US-DATA: Continuation of Ser. No. 383,056, Feb. 3, 1995, abandoned.

ABSTRACT:
Described is a polypeptide comprising one or more zinc fingers. The polypeptide binds to new polynucleotide subsites with high affinity and consequently has a binding specificity that differs from wild type zinc finger proteins. The binding occurs through contacts between certain amino acid residues of the zinc fingers and the nucleic acids of the subsites. The polypeptide sequence of at least one zinc finger differs from wild type zinc fingers, and the difference involves at least one amino acid residue that contacts the bases of the polynucleotide during binding.

17. 5,772,993, Jun. 30, 1998, Osteocalcin promoter-based toxic gene therapy for the treatment of calcified tumors and tissues; Leland W. K. Chung, et al., 424/93.6, 9.2; 435/71.2, 320.1; 514/44 [IMAGE AVAILABLE]

US PAT NO: 5,772,993 [IMAGE AVAILABLE] L4: 17 of 26

ABSTRACT:
A recombinant adenovirus Ad-OC-TK was constructed, with cell specific gene expression, which contains osteocalcin (OC) promoter that drives the expression of herpes simplex virus thymidine kinase (TK), the addition of acyclovir (ACV), a pro-drug for the inhibition of cell proliferation, to Ad-OC-TK resulted in the induction of osteoblast-specific cell death in vitro. The Ad-OC-TK virus plus ACV treatment is highly selective in blocking the growth of both murine and human osteosarcoma cell lines in vitro and murine osteosarcoma in vivo.

18. 5,747,469, May 5, 1998, Methods and compositions comprising DNA damaging agents and p53; Jack A. Roth, et al., 514/44; 435/320.1, 375; 514/2 [IMAGE AVAILABLE]

US PAT NO: 5,747,469 [IMAGE AVAILABLE] L4: 18 of 26
REL-US-DATA: Continuation-in-part of Ser. No. 145,826, Oct. 29, 1993, which is a continuation-in-part of Ser. No. 960,513, Oct. 13, 1992, which is a continuation-in-part of Ser. No. 665,538, Mar. 6, 1991, abandoned.

ABSTRACT:
The present invention relates to the use of tumor suppressor genes in combination with a DNA damaging agent or factor for use in killing cells, and in particular cancerous cells. A tumor suppressor gene, **p53**, was delivered via a recombinant **adenovirus**-mediated gene transfer both in vitro and in vivo, in combination with a chemotherapeutic agent. Treated cells underwent apoptosis with specific DNA fragmentation. Direct injection of the **p53**-**adenovirus** construct into tumors subcutaneously, followed by intraperitoneal administration of a DNA damaging agent, cisplatin, induced massive apoptotic destruction of the tumors. The invention also provides for the clinical application of a regimen combining gene replacement using replication-deficient wild-type **p53** **adenovirus** and DNA-damaging drugs for treatment of human cancer.

19. 5,716,830, Feb. 10, 1998, Human prostatic cell lines immortalized by adenovirus 12-simian virus 40 (AD12/SV40) hybrid virus; Mukta M. Webber, et al., 435/6, 29 [IMAGE AVAILABLE]

US PAT NO: 5,716,830 [IMAGE AVAILABLE] L4: 19 of 26
REL-US-DATA: Division of Ser. No. 234,981, Apr. 28, 1994, Pat. No. 5,610,043.

ABSTRACT:
Immortalized non-tumorigenic or tumorigenic human prostatic epithelial and fibroblast cell lines and derivatives thereof, containing DNA of a hybrid virus between adenovirus 12 and simian virus 40. The cell lines are useful for research on causes, treatment and prevention of prostate cancer, benign prostatic hyperplasia, male infertility, birth defects, aging and assessment of environmental toxic agents.

20. 5,707,618, Jan. 13, 1998, Adenovirus vectors for gene therapy; Donna Armentano, et al., 424/93.21, 93.2; 435/320.1; 514/44 [IMAGE AVAILABLE]

US PAT NO: 5,707,618 [IMAGE AVAILABLE] L4: 20 of 26

ABSTRACT:
The present invention relates to novel adenovirus vectors for use in gene therapy which are designed to prevent the generation of replication-competent adenovirus (RCA) during in vitro propagation and clinical use. The invention also provides methods for the production of the novel virus vectors. These vectors maximize safety for clinical applications in which adenovirus vectors are used to transfer genes into recipient cells for gene therapy.

21. 5,677,178, Oct. 14, 1997, Cytopathic viruses for therapy and prophylaxis of neoplasia; Francis McCormick, 435/325, 235.1, 375; 536/23.1, 24.1 [IMAGE AVAILABLE]

US PAT NO: 5,677,178 [IMAGE AVAILABLE] L4: 21 of 26
REL-US-DATA: Continuation of Ser. No. 198,184, Feb. 16, 1994, abandoned, which is a continuation-in-part of Ser. No. 17,525, Feb. 16, 1993, abandoned.

ABSTRACT:
Methods and compositions for treating neoplastic conditions by viral-based therapy are provided. Mutant virus lacking viral proteins which bind and/or inactivate p53 or RB are administered to a patient having a neoplasm which comprises cells lacking p53 and/or RB function. The mutant virus is able to substantially produce a replication phenotype in neoplastic cells but is substantially unable to produce a replication phenotype in non-replicating, non-neoplastic cells having essentially normal p53 and/or RB function. The preferential generation of replication phenotype in neoplastic cells results in a preferential killing of the neoplastic cells, either directly or by expression of a cytotoxic gene in cells expressing a viral replication phenotype.

22. 5,656,725, Aug. 12, 1997, Peptides and compositions which modulate apoptosis; Thomas D. Chittenden, et al., 530/324, 325, 326, 327, 328, 329, 330 [IMAGE AVAILABLE]

US PAT NO: 5,656,725 [IMAGE AVAILABLE] L4: 22 of 26

ABSTRACT:
The present invention is directed to novel peptides and compositions capable of modulating apoptosis in cells, and to methods of modulating apoptosis employing the novel peptides and compositions of the invention. In one aspect, the invention is directed to a novel peptide designated the "GD domain," which is essential both to Bak's interaction with Bcl-x.sub.L, and to Bak's cell killing function. Methods of identifying agonists or antagonists of GD domain function are provided. The GD domain is responsible for mediating key protein/protein interactions of significance to the actions of multiple cell death regulatory molecules.

23. 5,637,456, Jun. 10, 1997, Rapid test for determining the amount of functionally inactive gene in a gene therapy vector preparation; Jack A. Roth, et al., 435/5, 6, 320.1 [IMAGE AVAILABLE]

US PAT NO: 5,637,456 [IMAGE AVAILABLE] L4: 23 of 26

ABSTRACT:
The present invention relates generally to the area of quality control for recombinant agents to be used in gene therapy. Specifically, the invention concerns an assay used to identify the percentage of defective or therapeutically inactive vector in a vector stock.

24. 5,610,043, Mar. 11, 1997, Human prostatic cell lines immortalized by adenovirus 12-simian virus 40 (AD12/SV40) hybrid virus; Mukta M. Webber, et al., 435/467, 366, 371 [IMAGE AVAILABLE]

US PAT NO: 5,610,043 [IMAGE AVAILABLE] L4: 24 of 26

ABSTRACT:
Immortalized non-tumorigenic or tumorigenic human prostatic epithelial and fibroblast cell lines and derivatives thereof, containing DNA of a hybrid virus between adenovirus 12 and simian virus 40. The cell lines are useful for research on causes, treatment and prevention of prostate cancer, benign prostatic hyperplasia, male infertility, birth defects, aging and assessment of environmental toxic agents.

25. 5,604,113, Feb. 18, 1997, Cells having oncogene-suppressed p53-mediated apoptosis and methods of use to identify anti-oncogenic compounds; Eileen White, et al., 435/29, 325, 353 [IMAGE AVAILABLE]

US PAT NO: 5,604,113 [IMAGE AVAILABLE] L4: 25 of 26

ABSTRACT:
The present invention is a method for determining if a compound potentially modulates the ability of a putative oncogene to suppress p53-mediated effects, comprising (A) adding an amount of said compound to genetically engineered cells that express (i) a gene product that induces

p53 mediated apoptosis; (ii) a gene product for a p53 gene, wherein either the gene or the gene product are externally controllable; and (iii) a putative oncogene that inhibits the effect of the gene product that induces p53 mediated apoptosis, and (B) examining said cells to determine whether apoptosis has occurred. In particular, the invention provides a method wherein said gene product that induces **p53** mediated apoptosis is **adenovirus** gene E1A, wherein said externally controllable p53 gene product is a temperature-sensitive mutant of p53, and wherein the putative oncogene is selected from the group bcl-2, ras, or the adenovirus E1B(19K) gene. In addition, the invention provides genetically engineered cell lines that can be used to carry out the methods described.

26. 5,569,824, Oct. 29, 1996, Transgenic mice containing a disrupted p53 gene; Lawrence A. Donehower, et al., 800/10; 424/9.1; 800/18 [IMAGE AVAILABLE]

US PAT NO: 5,569,824 [IMAGE AVAILABLE] L4: 26 of 26
REL-US-DATA: Continuation of Ser. No. 816,740, Jan. 3, 1992, abandoned, which is a continuation-in-part of Ser. No. 637,563, Jan. 4, 1991, abandoned.

ABSTRACT:
A desired non-human animal or an animal cell or human cell which contains a predefined, specific and desired alteration in at least one of its two p53 chromosomal alleles, such that at least one of these alleles contains a mutation which alters the expression of the allele, and the other of the alleles expresses either a normal p53 gene product, or comprises an identical or different p53 mutation.

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Search Results - Record(s) 1 through 33 of 33 returned.

1. Document ID: US 5976786 A
Entry 1 of 33
File: USPT
Nov 2, 1999

US-PAT-NO: 5976786
DOCUMENT-IDENTIFIER: US 5976786 A

TITLE: Screening methods for the identification of compounds that modulate apoptosis in
immunodeficiency virus infected cells

DATE-ISSUED: November 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Finkel; Terri H.	Englewood	CO	N/A	N/A
Casella; Carolyn	Denver	CO	N/A	N/A

US-CL-CURRENT: 435/5; 435/375

ABSTRACT:

Disclosed is a method to limit infection by an immunodeficiency virus. The method includes
inhibiting an immunodeficiency virus protein which regulates apoptosis in cells. Also disclosed
are methods to identify compounds that regulate cellular inhibitors of apoptosis in cells infected
with an immunodeficiency virus and compounds identified thereby.
7 Claims, 2 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 2

2. Document ID: US 5968824 A
Entry 2 of 33
File: USPT
Oct 19, 1999

US-PAT-NO: 5968824
DOCUMENT-IDENTIFIER: US 5968824 A

TITLE: Agents for inducing apoptosis and applications of said agents in therapy

DATE-ISSUED: October 19, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Spruce; Barbara Ann	Rait, Perthshire PH2 7SB		N/A	GBX
Prescott; Alan	Kingskettle, Fife KY15 7PY		N/A	GBX
Bottger; Angelika	Newport-on-tay DD6 8JJ		N/A	GBX
Dewar; Deborah Ann	Dundee DD1 7RR		N/A	GBX

US-CL-CURRENT: 435/375; 424/427, 427/2.24, 435/377, 514/279, 514/408, 514/912, 514/954

ABSTRACT:

Agents which modulate pathways of apoptotic induction or repression in which products of opioid
peptide precursors genes participate, useful as inducers of apoptosis in cells and in tumor cells
in particular, are disclosed. Methods of treatment employing such agents and pharmaceutical
compositions containing them are also described.
6 Claims, 6 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

3. Document ID: US 5968821 A
Entry 3 of 33
File: USPT
Oct 19, 1999

US-PAT-NO: 5968821
DOCUMENT-IDENTIFIER: US 5968821 A

TITLE: Cell-cycle regulatory proteins, and uses related thereto

DATE-ISSUED: October 19, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Beach; David H.	Huntington Bay	NY	N/A	N/A
Demetrick; Douglas J.	E. Northport	NY	N/A	N/A
Serrano; Manuel	Mill Neck	NY	N/A	N/A
Hannon; Gregory J.	Huntington	NY	N/A	N/A

US-CL-CURRENT: 435/325; 435/320.1, 435/455, 435/6, 435/69.1, 536/23.1

ABSTRACT:

The present invention relates to the discovery in eukaryotic cells, particularly mammalian cells,
of a novel family of cell-cycle regulatory proteins ("CCR-proteins"). As described herein, this
family of proteins includes a polypeptide having an apparent molecular weight of 16 kDa, and a
polypeptide having an apparent molecular weight of approximately 15 kDa, each of which can
function as an inhibitor of cell-cycle progression, and therefore ultimately of cell growth. Thus,
similar to the role of p21 to the p53 checkpoint, the subject CCR-proteins may function
coordinately with the cell-cycle regulatory protein, retinoblastoma (RB). Furthermore, the
CCR-protein family includes a protein having an apparent molecular weight of 13.5 kDa (hereinafter
"p13.5"). The presumptive role of p13.5, like p16 and p15, is in the regulation of the cell-cycle.

35 Claims, 11 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 11

4. Document ID: US 5962316 A
Entry 4 of 33
File: USPT
Oct 5, 1999

US-PAT-NO: 5962316
DOCUMENT-IDENTIFIER: US 5962316 A

TITLE: Cell-cycle regulatory proteins, and uses related thereto

DATE-ISSUED: October 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Beach; David H.	Huntington Bay	NY	N/A	N/A
Demetrick; Douglas J.	Northport	NY	N/A	N/A
Serrano; Manuel	Mill Neck	NY	N/A	N/A
Hannon; Gregory J.	Huntington	NY	N/A	N/A

US-CL-CURRENT: 435/325; 424/185.1, 424/93.21, 435/320.1, 435/455, 435/6, 435/69.1, 514/44,
530/350, 536/23.1, 536/23.4, 536/23.5, 536/24.1

ABSTRACT:

The present invention relates to the discovery in eukaryotic cells, particularly mammalian cells,
of a novel family of cell-cycle regulatory proteins ("CCR-proteins"). As described herein, this
family of proteins includes a polypeptide having an apparent molecular weight of 16 kDa, and a
polypeptide having an apparent molecular weight of approximately 15 kDa, each of which can
function as an inhibitor of cell-cycle progression, and therefore ultimately of cell growth. Thus,
similar to the role of p21 to the p53 checkpoint, the subject CCR-proteins may function
coordinately with the cell-cycle regulatory protein, retinoblastoma (RB). Furthermore, the
CCR-protein family includes a protein having an apparent molecular weight of 13.5 kDa (hereinafter
"p13.5"). The presumptive role of p13.5, like p16 and p15, is in the regulation of the cell-cycle.

40 Claims, 11 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 11

5. Document ID: US 5959081 A
Entry 5 of 33
File: USPT
Sep 28, 1999

US-PAT-NO: 5959081
DOCUMENT-IDENTIFIER: US 5959081 A

TITLE: Zinc binding LIM protein S2-6

DATE-ISSUED: September 28, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lecka-Czernik; Beata	Little Rock	AR	72227	N/A

US-CL-CURRENT: 530/358; 435/320.1, 435/325, 536/23.1

ABSTRACT:

A substantially pure S2-6 protein (a) having a zinc binding LIM domain, (b) whose mRNA is
preferentially expressed in nonproliferating or growth inhibited human diploid fibroblasts, (c)
whose mRNA is overexpressed in senescent human diploid fibroblasts or human diploid fibroblasts
derived from a patient with Werner Syndrome, and (c) whose mRNA expression is reduced or abolished
in fetal human diploid fibroblasts, immortalized cells, cancerous cells and other highly
proliferative cells.
8 Claims, 7 Drawing figures
Exemplary Claim Number: 1,4
Number of Drawing Sheets: 9

6. Document ID: US 5955429 A
Entry 6 of 33
File: USPT
Sep 21, 1999

US-PAT-NO: 5955429

DOCUMENT-IDENTIFIER: US 5955429 A

TITLE: Human apoptosis-associated protein

DATE-ISSUED: September 21, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hillman; Jennifer L.	San Jose	CA	N/A	N/A
Goli; Surya K.	Sunnyvale	CA	N/A	N/A

US-CL-CURRENT: 514/12; 530/350

ABSTRACT:

The present invention provides a novel human apoptosis-associated protein (NHAAP) and polynucleotides which identify and encode NHAAP. The invention also provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding NHAAP and a method for producing NHAAP. The invention also provides for agonists, antibodies, or antagonists specifically binding NHAAP, and their use, in the prevention and treatment of diseases associated with expression of NHAAP. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding NHAAP for the treatment of diseases associated with the expression of NHAAP. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding NHAAP.

3 Claims, 3 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

7. Document ID: US 5905146 A
Entry 7 of 33
File: USPT
May 18, 1999

US-PAT-NO: 5905146
DOCUMENT-IDENTIFIER: US 5905146 A

TITLE: DNA binding protein S1-3

DATE-ISSUED: May 18, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lecka-Czernik; Beata	Little Rock	AR	N/A	N/A

US-CL-CURRENT: 536/23.5; 435/252.3, 435/320.1, 435/325, 536/24.31

ABSTRACT:

A substantially pure S1-3 protein (a) being a DNA binding protein containing three zinc finger domains, (b) whose mRNA is overexpressed in senescent human diploid fibroblasts or human diploid fibroblasts derived from a patient with Werner Syndrome, and (c) whose mRNA is not expressed in fetal human diploid fibroblasts.
10 Claims, 14 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 19

8. Document ID: US 5889169 A
Entry 8 of 33
File: USPT
Mar 30, 1999

US-PAT-NO: 5889169
DOCUMENT-IDENTIFIER: US 5889169 A

TITLE: Cell cycle regulatory protein p16 gene

DATE-ISSUED: March 30, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Beach; David H.	Huntington Bay	NY	N/A	N/A
Demetrick; Douglas J.	E. Northport	NY	N/A	N/A
Serrano; Manuel	Mill Neck	NY	N/A	N/A
Hannon; Gregory J.	Huntington	NY	N/A	N/A
Quelle; Dawn E.	Cordova	TN	N/A	N/A
Sherr; Charles J.	Memphis	TN	N/A	N/A

US-CL-CURRENT: 536/23.5; 530/358, 536/23.7, 536/23.74

ABSTRACT:

The present invention relates to the discovery in eukaryotic cells, particularly mammalian cells, of a novel family of cell-cycle regulatory proteins ("CCR-proteins"). As described herein, these family of proteins includes a polypeptide having an apparent molecular weight of 16 kDa

(hereinafter "p16.sup.INK4 " OR "p16") and which can function as an inhibitor of cell-cycle
progression, and therefore ultimately of cell growth, and that similar to role of p21 and p53, the
p16 protein may function coordinately with the cell cycle regulatory protein, retinoblastoma (Rb).
Furthermore, the CCR-protein family includes a protein having an apparent molecular weight of 13.5
kDa (hereinafter "p13.5"). The presumptive role of p13.5, like p16, is in the regulation of the
cell-cycle.
29 Claims, 10 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 10

9. Document ID: US 5886149 A
Entry 9 of 33
File: USPT
Mar 23, 1999

US-PAT-NO: 5886149
DOCUMENT-IDENTIFIER: US 5886149 A

TITLE: P53 response genes

DATE-ISSUED: March 23, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Buckbinder; Leonard	Doylestown	PA	N/A	N/A
Talbott; Randy	Freehold	NJ	N/A	N/A
Seizinger; Bernd R.	Stockton	NJ	N/A	N/A
Kley; Nikolai	Princeton Junction	NJ	N/A	N/A

US-CL-CURRENT: 530/350; 536/23.5

ABSTRACT:

This present invention concerns polypeptide molecules comprising human p53 response protein
PIGI-1.
3 Claims, 13 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 11

10. Document ID: US 5882880 A
Entry 10 of 33
File: USPT
Mar 16, 1999

US-PAT-NO: 5882880

DOCUMENT-IDENTIFIER: US 5882880 A

TITLE: Human checkpoint gene and gene for antisense RNA thereof

DATE-ISSUED: March 16, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Canaani; Dan	Raanana	N/A	N/A	ILX

US-CL-CURRENT: 435/29; 435/320.1, 530/350, 536/23.5

ABSTRACT:

A novel human radiation protecting checkpoint (RAP-1) gene and the RAP-1 protein encoded by the
gene are disclosed. RAP-1 is believed to be involved in the regulation of cell cycle progression
and/or programmed cell death (apoptosis). A human antisense RNA which can bind to the mRNA of the
human RAP-1 protein, as well as the DNA from which the antisense RNA is transcribed are also
disclosed. Additionally, a method for isolating DNA damage-monitoring checkpoint genes is
described. The use of the DNA and RNA sequences and of the protein of the invention for the eary
detection, prevention and/or treatment of cancer, AIDS and other diseases is also discussed.
13 Claims, 13 Drawing figures
Exemplary Claim Number: 10
Number of Drawing Sheets: 8

11. Document ID: US 5863904 A
Entry 11 of 33
File: USPT
Jan 26, 1999

US-PAT-NO: 5863904
DOCUMENT-IDENTIFIER: US 5863904 A

TITLE: Methods for treating cancers and restenosis with P21

DATE-ISSUED: January 26, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nabel; Gary J.	Ann Arbor	MI	N/A	N/A
Yang; Zhi-yong	Ann Arbor	MI	N/A	N/A
Nabel; Elizabeth G.	Ann Arbor	MI	N/A	N/A

US-CL-CURRENT: 514/44; 435/375, 435/69.1

ABSTRACT:

The p21 gene encodes a cyclin dependent kinase inhibitor which affects cell cycle progression, but
the role of this gene product in altering tumor growth has not been established. The present
inventors have now discovered that the growth of malignant cells in vivo is inhibited by
expression of p21. Expression of p21 resulted in an accumulation of cells in G.sub.0 /G.sub.1,
alteration in morphology, and cell differentiation.
8 Claims, 14 Drawing figures
Exemplary Claim Number: 1,5
Number of Drawing Sheets: 11

12. Document ID: US 5861249 A
Entry 12 of 33
File: USPT
Jan 19, 1999

US-PAT-NO: 5861249
DOCUMENT-IDENTIFIER: US 5861249 A

TITLE: Assays and reagents for identifying modulators of cdc25-mediated mitotic activation

DATE-ISSUED: January 19, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Beach; David H.	Huntington Bay	NY	N/A	N/A
Galaktionov; Konstantin	Cold Spring Harbor	NY	N/A	N/A

US-CL-CURRENT: 435/6; 435/7.1

ABSTRACT:

The present invention makes available assays and reagents for identifying agents which can be used
to modulate at least one proliferation, differentiation and cell death by apoptosis.
10 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

13. Document ID: US 5858679 A
Entry 13 of 33
File: USPT
Jan 12, 1999

US-PAT-NO: 5858679
DOCUMENT-IDENTIFIER: US 5858679 A

TITLE: Method for determining the presence of functional p53 by measuring GADD45 protein
expression

DATE-ISSUED: January 12, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fornace, Jr.; Albert J.	Bethesda	MD	20817	N/A
Kastan; Michael B.	Owings Mill	MD	21117	N/A
Carrier; France	Bethesda	MD	20814-201	N/A

8

US-CL-CURRENT: 435/7.1; 530/386

ABSTRACT:

The dependence of ionizing radiation-induced GADD45 mRNA and protein expression on the presence of
functional p53 in mammalian cells is disclosed. First and second oligonucleotide sequences are
provided which can form a double-stranded oligomer capable of binding to functional p53 protein.
The present invention demonstrates that the dependence of ionizing radiation-induced GADD45 mRNA
and protein expression on the presence of functional p53 and the binding of functional p53 to a
double-stranded oligomer binding sequence can serve as the bases for methods for determining the
presence of functional p53 in mammalian cell lines and tumors.
6 Claims, 8 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

14. Document ID: US 5858715 A
Entry 14 of 33
File: USPT
Jan 12, 1999

US-PAT-NO: 5858715
DOCUMENT-IDENTIFIER: US 5858715 A

TITLE: Human apoptosis-associated protein

DATE-ISSUED: January 12, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hillman; Jennifer L.	San Jose	CA	N/A	N/A
Goli; Surya K.	Sunnyvale	CA	N/A	N/A

US-CL-CURRENT: 435/69.1; 435/252.3, 435/254.11, 435/320.1, 435/325, 536/23.5

ABSTRACT:

The present invention provides a novel human apoptosis-associated protein (NHAAP) and polynucleotides which identify and encode NHAAP. The invention also provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding NHAAP and a method for producing NHAAP. The invention also provides for agonists, antibodies, or antagonists specifically binding NHAAP, and their use, in the prevention and treatment of diseases associated with expression of NHAAP. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding NHAAP for the treatment of diseases associated with the expression of NHAAP. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding NHAAP.

8 Claims, 3 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

15. Document ID: US 5821051 A
Entry 15 of 33
File: USPT
Oct 13, 1998

US-PAT-NO: 5821051
DOCUMENT-IDENTIFIER: US 5821051 A

TITLE: E6 binding proteins

DATE-ISSUED: October 13, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Androphy; Elliot	Natick	MA	N/A	N/A
Chen; Jason J.	Boston	MA	N/A	N/A

US-CL-CURRENT: 435/5; 530/350, 530/352, 530/357, 530/358

ABSTRACT:

An assay for screening test compounds to indentify agents which modulate the binding of an E6-BP polypeptide with a papilloma virus E6 protein. The assay includes combining, as a cell-free system, an E6-binding protein or fragment thereof which binds to the E6 protein, and a test compound, and detecting the formation of a complex which includes the E6 protein and the E6-binding protein. A change in the formation of the complex in the presence of the test compound is indicative of an agent that modulates interaction between an E6 and an E6-binding protein.

31 Claims, 1 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 1

16. Document ID: US 5840579 A
Entry 16 of 33
File: USPT
Nov 24, 1998

US-PAT-NO: 5840579
DOCUMENT-IDENTIFIER: US 5840579 A

TITLE: Nucleic acids encoding p53 mutations which suppress p53 cancer mutations

DATE-ISSUED: November 24, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Boeke; Jef D.	Baltimore	MD	N/A	N/A
Brachmann; Rainer K.	Baltimore	MD	N/A	N/A

US-CL-CURRENT: 435/325; 435/254.2, 435/320.1, 536/23.1

ABSTRACT:

Intragenic suppressor mutations of common p53 mutations are able to function in cis and/or trans. These mutations are useful for identifying small molecule drugs which function in a similar fashion. In addition, the mutations themselves may be useful therapeutically, especially if they function in trans. Methods for rapidly obtaining this type of mutant employ a yeast selection system. Cells having both the negative mutation and intragenic suppressor are useful for studying the interactions of the two, in particular in determining the structure of the homotetramers and heterotetramers.

32 Claims, 12 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 10

17. Document ID: US 5847083 A
Entry 17 of 33
File: USPT
Dec 8, 1998

US-PAT-NO: 5847083
DOCUMENT-IDENTIFIER: US 5847083 A

TITLE: Modified p53 constructs which enhance DNA binding

DATE-ISSUED: December 8, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE

ZIP CODE
COUNTRY
Halazonetis; Thanos D.
Philadelphia
PA
N/A
N/A

US-CL-CURRENT: 530/358; 435/320.1, 435/69.1, 536/23.4, 536/23.5

ABSTRACT:

A modified p53 protein or peptide having DNA binding in which amino acid residue 284 of a p53
protein or protein fragment is changed to Arginine or Lysine, is described. Also described are
nucleotide sequences encoding the modified protein and vectors capable of expressing it.
18 Claims, 5 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

18. Document ID: US 5843659 A
Entry 18 of 33
File: USPT
Dec 1, 1998

US-PAT-NO: 5843659
DOCUMENT-IDENTIFIER: US 5843659 A

TITLE: Apoptosis gene EI24, compositions, and methods of use

DATE-ISSUED: December 1, 1998

INVENTOR-INFORMATION:
NAME
CITY
STATE
ZIP CODE
COUNTRY
Lehar; Sophie M.
Berlin
MA
N/A
N/A
Guild; Braydon C.
Concord
MA
N/A
N/A

US-CL-CURRENT: 435/6; 435/320.1, 435/325, 435/69.1, 435/91.4, 536/23.5

ABSTRACT:

Disclosed is the isolation and characterization of EI24, a novel gene whose 2.4 kb mRNA is induced
following etoposide treatment. Induction of EI24 mRNA by etoposide required expression of
wild-type p53. Overexpression of functional p53 was sufficient to induce expression of the EI24
mRNA. The EI24 mRNA was also induced in a p53-dependent manner by ionizing irradiation of primary
murine thymocytes. The invention is thus directed to an isolated EI24 protein, nucleotide
sequences coding for and regulating expression of the protein, antibodies directed against the
protein, and recombinant vectors and host cells containing the genetic sequences coding for and
regulating the expression of the protein sequence. The invention is also directed to genomic DNA,
cDNA, and RNA encoding the EI24 protein sequence and to corresponding antisense RNA sequences.
Antibodies can be used to detect EI24 in biological specimens, including, for example, human
tissue samples. The present invention is further directed to methods of treating degenerative
disorders characterized in inappropriate cell proliferation or inappropriate cell death. The
present invention is further directed to methods for diagnosing degenerative disorders
characterized in inappropriate cell proliferation or inappropriate cell death, as well as methods
for monitoring the progress of such degenerative disorders.
18 Claims, 9 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

19. Document ID: US 5843654 A
Entry 19 of 33
File: USPT
Dec 1, 1998

US-PAT-NO: 5843654
DOCUMENT-IDENTIFIER: US 5843654 A

TITLE: Rapid detection of mutations in the p53 gene

DATE-ISSUED: December 1, 1998

INVENTOR-INFORMATION:
NAME
CITY
STATE
ZIP CODE
COUNTRY
Heisler; Laura M.
Madison
WI
N/A
N/A
Fors; Lance
Monrovia
CA
N/A
N/A
Brow; Mary Ann D.
Madison
WI
N/A
N/A

US-CL-CURRENT: 435/6; 435/194, 435/91.1

ABSTRACT:

The present invention relates to means for cleaving a nucleic acid cleavage structure in a
site-specific manner. Enzymes, including 5' nucleases and 3' exonucleases, are used to screen for
known and unknown mutations, including single base changes, in the human p53 gene. Methods are
provided which allow for the identification of genetic mutations in the human p53 gene in a
sample.
25 Claims, 118 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 79

20. Document ID: US 5834487 A
Entry 20 of 33
File: USPT
Nov 10, 1998

US-PAT-NO: 5834487
DOCUMENT-IDENTIFIER: US 5834487 A

TITLE: Inhibition of 26S and 20S proteasome by indanones

DATE-ISSUED: November 10, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lum; Robert T.	Palo Alto	CA	N/A	N/A
Schow; Steven R.	Redwood City	CA	N/A	N/A
Joly; Alison	San Mateo	CA	N/A	N/A
Kerwar; Suresh	Westchester	NY	N/A	N/A
Nelson; Marek G.	Sunol	CA	N/A	N/A
Wick; Michael M.	Chestnut Hill	MA	N/A	N/A

US-CL-CURRENT: 514/319; 514/19, 514/561, 514/677

ABSTRACT:

This invention is a method for inhibiting cell proliferation using indanones.
11 Claims, 3 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

21. Document ID: US 5747650 A
Entry 21 of 33
File: USPT
May 5, 1998

US-PAT-NO: 5747650
DOCUMENT-IDENTIFIER: US 5747650 A

TITLE: P53AS protein and antibody therefor

DATE-ISSUED: May 5, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kulesz-Martin; Molly F.	Buffalo	NY	N/A	N/A

US-CL-CURRENT: 530/387.7; 530/387.1, 530/388.8, 530/389.1, 530/389.2

ABSTRACT:

In accordance with the present invention, we have discovered and purified a protein designated
herein as p53as, which protein is present in normal cells of a mammal and is essentially identical
to known normal growth controlling protein p53 of the same mammal, at least until the final 50
amino acids of the carboxy terminal end of the protein. The invention further includes an antibody
specific for protein p53as, which antibody is designated herein as Ab p53as. The antibody may be
either a monoclonal or polyclonal antibody and may be specific for p53as of any particular mammal
such as mice and humans.
11 Claims, 26 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 11

22. Document ID: US 5747469 A
Entry 22 of 33
File: USPT
May 5, 1998

US-PAT-NO: 5747469
DOCUMENT-IDENTIFIER: US 5747469 A

TITLE: Methods and compositions comprising DNA damaging agents and p53

DATE-ISSUED: May 5, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Roth; Jack A.	Houston	TX	N/A	N/A
Fujiwara; Toshiyoshi	Okayama	N/A	N/A	JPX
Grimm; Elizabeth A.	Houston	TX	N/A	N/A
Mukhopadhyay; Tapas	Houston	TX	N/A	N/A
Zhang; Wei-Wei	Houston	TX	N/A	N/A
Owen-Schaub; Laurie B.	Houston	TX	N/A	N/A

US-CL-CURRENT: 514/44; 435/320.1, 435/375, 514/2

ABSTRACT:

The present invention relates to the use of tumor suppressor genes in combination with a DNA damaging agent or factor for use in killing cells, and in particular cancerous cells. A tumor suppressor gene, p53, was delivered via a recombinant adenovirus-mediated gene transfer both in vitro and in vivo, in combination with a chemotherapeutic agent. Treated cells underwent apoptosis with specific DNA fragmentation. Direct injection of the p53-adenovirus construct into tumors subcutaneously, followed by intraperitoneal administration of a DNA damaging agent, cisplatin, induced massive apoptotic destruction of the tumors. The invention also provides for the clinical application of a regimen combining gene replacement using replication-deficient wild-type p53 adenovirus and DNA-damaging drugs for treatment of human cancer.
105 Claims, 45 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 22

23. Document ID: US 5733920 A
Entry 23 of 33
File: USPT
Mar 31, 1998

US-PAT-NO: 5733920
DOCUMENT-IDENTIFIER: US 5733920 A

TITLE: Inhibitors of cyclin dependent kinases

DATE-ISSUED: March 31, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mansuri; Muzammil M.	Lexington	MA	N/A	N/A
Murthi; Krishna K.	Waltham	MA	N/A	N/A
Pal; Kollol	Needham	MA	N/A	N/A

US-CL-CURRENT: 514/337; 514/254, 514/256, 514/274, 514/312, 514/320, 514/324, 514/432 , 514/456, 544/238, 544/315, 544/318, 544/408, 546/153, 546/196, 546/283.1, 549/23, 549/401, 549/403

ABSTRACT:

The invention provides novel inhibitors of cyclin-dependent kinases, in particular inhibitors of the CDK/cyclin complexes such as CDK4/cyclin D1. The novel compounds are analogs of chromones. These compounds can be used for inhibiting excessive or abnormal cell proliferation. Thus, the novel compounds are useful for treating a subject with a disorder associated with excessive cell proliferation, such as cancer.
37 Claims, 3 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 2

24. Document ID: US 5721340 A
Entry 24 of 33
File: USPT
Feb 24, 1998

US-PAT-NO: 5721340
DOCUMENT-IDENTIFIER: US 5721340 A

TITLE: p53 proteins with altered tetramerization domains

DATE-ISSUED: February 24, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Halazonetis; Thanos D.	Philadelphia	PA	N/A	N/A

US-CL-CURRENT: 530/350; 435/320.1, 435/69.7, 435/7.1, 530/352, 530/358, 536/23.1

ABSTRACT:

The present invention provides p53 proteins with altered tetramerization domains that retain wild-type p53 function, and the ability to form tetramers and have at least one of the following characteristics: (1) do not hetero-oligomerize with wild-type p53 or tumor-derived p53 mutants, and (2) restricted DNA binding specificity from an alteration in the way that the tetramerization domain orients the DNA binding domains of a p53 tetramer relative to one another. The invention also provides nucleic acids encoding the above proteins and methods of enhancing the cellular response to DNA damaging agents, treating diseases characterized by abnormal cell proliferation, and inducing immune tolerance to facilitate transplants and treatment of autoimmune disease, by administration of proteins of the invention or nucleic acid sequences encoding the proteins of the invention.
27 Claims, 25 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 5

25. Document ID: US 5693617 A
Entry 25 of 33
File: USPT
Dec 2, 1997

US-PAT-NO: 5693617
DOCUMENT-IDENTIFIER: US 5693617 A

TITLE: Inhibitors of the 26s proteolytic complex and the 20s proteasome contained therein

DATE-ISSUED: December 2, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Stein; Ross L.	Sudbury	MA	N/A	N/A
Ma; Yu-Ting	Needham	MA	N/A	N/A
Brand; Stephen	Lincoln	MA	N/A	N/A

US-CL-CURRENT: 514/18; 514/19, 530/331, 560/159, 560/20, 560/27, 560/31, 560/32, 560/41, 560/47

ABSTRACT:

Disclosed herein is a method for reducing the rate of degradation of proteins in an animal comprising contacting cells of the animal with certain proteasome inhibitors. The structure of the inhibitors are also disclosed.

22 Claims, 10 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 8

26. Document ID: US 5672508 A
Entry 26 of 33
File: USPT
Sep 30, 1997

US-PAT-NO: 5672508
DOCUMENT-IDENTIFIER: US 5672508 A

TITLE: Inhibitors of cell-cycle progression, and uses related thereto

DATE-ISSUED: September 30, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gyuris; Jeno	Winchester	MA	N/A	N/A
Lamphere; Lou	Boston	MA	N/A	N/A
Beach; David	Huntington Bay	NY	N/A	N/A

US-CL-CURRENT: 435/320.1; 536/23.4, 536/23.5

ABSTRACT:

The present invention pertains to novel inhibitors of cyclin-dependent kinases (CDKs), particularly CDK/cyclin complexes, which inhibitors can be used to control proliferation and/or differentiation of cells in which the inhibitors are introduced. More specifically, the inhibitors of the invention are chimeric proteins which include CDK-binding motifs from two or more different proteins. For example, the subject chimeric proteins can be generated from the in-frame fusion of coding sequences from two different CDK inhibitor proteins, such as may be derived from fusion of coding sequences for an INK4 protein and coding sequences for a CIP protein. Chimeric proteins of the present invention have been observed to be more potent inhibitors of cyclin/CDK complexes than were either of the portions of the chimeric protein individually.

37 Claims, 0 Drawing figures
Exemplary Claim Number: 1

27. Document ID: US 5667987 A
Entry 27 of 33
File: USPT
Sep 16, 1997

US-PAT-NO: 5667987
DOCUMENT-IDENTIFIER: US 5667987 A

TITLE: P53 response genes

DATE-ISSUED: September 16, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Buckbinder; Leonard	Doylestown	PA	N/A	N/A
Talbott; Randy	Freehold	NJ	N/A	N/A
Seizinger; Bernd R.	Stockton	NJ	N/A	N/A
Kley; Nikolai	Princeton Junction	NJ	N/A	N/A

US-CL-CURRENT: 435/69.1; 435/252.3, 435/254.11, 435/320.1, 536/23.5

ABSTRACT:

Nucleic acid sequences, particularly DNA sequences, coding for all or part of p53 response protein PIGI-1, expression vectors containing the DNA sequences, host cells containing the expression vectors, and methods utilizing these materials are disclosed. The invention also concerns polypeptide molecules comprising all or part of p53 response protein PIGI-1, and methods for producing these polypeptide molecules.

22 Claims, 13 Drawing figures
Exemplary Claim Number: 1

Number of Drawing Sheets: 13

28. Document ID: US 5616463 A
Entry 28 of 33
File: USPT
Apr 1, 1997

US-PAT-NO: 5616463
DOCUMENT-IDENTIFIER: US 5616463 A

TITLE: Methods for determining the presence of functional p53 in mammalian cells

DATE-ISSUED: April 1, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fornace, Jr.; Albert J.	Bethesda	MD	N/A	N/A
Kastan; Michael B.	Owings Mill	MD	N/A	N/A

US-CL-CURRENT: 435/6; 536/23.5

ABSTRACT:

The dependence of ionizing radiation-induced GADD45 mRNA expression on the presence of functional p53 in mammalian cells is disclosed. First and second oligonucleotide sequences are provided which can form a double-stranded oligomer capable of binding to functional p53 protein. The present invention demonstrates that the dependence of ionizing radiation-induced GADD45 mRNA expression on the presence of functional p53 and the binding of functional p53 to a double-stranded oligomer binding sequence can serve as the basis for methods for determining the presence of functional p53 in mammalian cell lines and tumors.
12 Claims, 5 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

29. Document ID: US 5604113 A
Entry 29 of 33
File: USPT
Feb 18, 1997

US-PAT-NO: 5604113
DOCUMENT-IDENTIFIER: US 5604113 A

TITLE: Cells having oncogene-suppressed p53-mediated apoptosis and methods of use to identify anti-oncogenic compounds

DATE-ISSUED: February 18, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
White; Eileen	Belle Mead	NJ	N/A	N/A
Chiou; Shuin-Kwei	N. Potomac	MD	N/A	N/A
Lin; Huey-Jen L.	Princeton	NJ	N/A	N/A

US-CL-CURRENT: 435/29; 435/325, 435/353

ABSTRACT:

The present invention is a method for determining if a compound potentially modulates the ability of a putative oncogene to suppress p53-mediated effects, comprising (A) adding an amount of said compound to genetically engineered cells that express (i) a gene product that induces p53 mediated apoptosis; (ii) a gene product for a p53 gene, wherein either the gene or the gene product are externally controllable; and (iii) a putative oncogene that inhibits the effect of the gene product that induces p53 mediated apoptosis, and (B) examining said cells to determine whether apoptosis has occurred. In particular, the invention provides a method wherein said gene product that induces p53 mediated apoptosis is adenovirus gene E1A, wherein said externally controllable p53 gene product is a temperature-sensitive mutant of p53, and wherein the putative oncogene is selected from the group bcl-2, ras, or the adenovirus E1B(19K) gene. In addition, the invention provides genetically engineered cell lines that can be used to carry out the methods described.
10 Claims, 13 Drawing figures
Exemplary Claim Number: 4
Number of Drawing Sheets: 7

30. Document ID: US 5573925 A
Entry 30 of 33
File: USPT
Nov 12, 1996

US-PAT-NO: 5573925
DOCUMENT-IDENTIFIER: US 5573925 A

TITLE: P53 proteins with altered tetramerization domains

DATE-ISSUED: November 12, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Halazonetis; Thanos D.	Philadelphia	PA	N/A	N/A

US-CL-CURRENT: 435/69.7; 514/44, 530/350, 536/23.4

ABSTRACT:

The present invention provides p53 proteins with altered tetramerization domains that retain wild-type p53 function, and the ability to form tetramers and have at least one of the following characteristics: (1) do not hetero-oligomerize with wild-type p53 or tumor-derived p53 mutants, and (2) restricted DNA binding specificity from an alteration in the way that the tetramerization domain orients the DNA binding domains of a p53 tetramer relative to one another. The invention also provides nucleic acids encoding the above proteins and methods of enhancing the cellular response to DNA damaging agents, treating diseases characterized by abnormal cell proliferation, and inducing immune tolerance to facilitate transplants and treatment of autoimmune disease, by administration of proteins of the invention or nucleic acid sequences encoding the proteins of the invention.

15 Claims, 24 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

31. Document ID: US 5525482 A
Entry 31 of 33
File: USPT
Jun 11, 1996

US-PAT-NO: 5525482
DOCUMENT-IDENTIFIER: US 5525482 A

TITLE: Method and cell line for testing cytotoxicity and mutagenicity of a chemical

DATE-ISSUED: June 11, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
States; J. Christopher	Royal Oak	MI	N/A	N/A
Hines; Ronald N.	West Bloomfield	MI	N/A	N/A
Novak; Raymond F.	West Bloomfield	MI	N/A	N/A

US-CL-CURRENT: 435/32; 435/29, 435/455

ABSTRACT:

A method of testing the cytotoxicity and mutagenicity of a chemical includes the steps of exposing test cells to the chemical in vitro, intracellularly metabolizing the chemical into a mutagenic or cytotoxic metabolite and then detecting gene/protein/cell damage in the test cells as an indication of the mutagenicity/cytotoxicity of the chemical.

A cell line is provided for testing cytotoxicity and mutagenicity of the chemicals, the cell line consisting essentially of fibroblasts normally having no detectable cytochrome P450 mixed function oxidase enzyme activity. The fibroblasts are transformed with chimeric gene constructs containing cytochrome P450 coding sequences and have intracellular cytochrome P450 oxidative metabolizing activity.

7 Claims, 16 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 12

32. Document ID: US 5180666 A
Entry 32 of 33
File: USPT
Jan 19, 1993

US-PAT-NO: 5180666
DOCUMENT-IDENTIFIER: US 5180666 A

TITLE: Method and cell line for testing mutagenicity of a chemical

DATE-ISSUED: January 19, 1993

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
States; J. Christopher	Royal Oak	MI	N/A	N/A
Hines; Ronald N.	West Bloomfield	MI	N/A	N/A
Novak; Raymond F.	West Bloomfield	MI	N/A	N/A

US-CL-CURRENT: 435/29; 435/366, 435/455

ABSTRACT:

A method of testing the mutagenicity of a chemical includes the steps of exposing test cells to the chemical in vitro, intracellularly metabolizing the chemical into a mutagenic or cytotoxic metabolite and then detecting gene/protein/cell damage in the test cells as an indication of the mutagenicity/cytotoxicity of the chemical.

A cell line is provided for testing mutagenicity of the chemicals, the cell line consisting essentially of fibroblasts normally having no detectable cytochrome P450 mixed function oxidase enzyme activity. The fibroblasts are transformed with chimeric gene constructs containing cytochrome P450 coding sequences and have intracellular cytochrome P450 oxidative metaboli zing activity.

15 Claims, 8 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 10

33. Document ID: AU 9890237 A, WO 9910486 A2

Entry 33 of 33

File: DWPI

Mar 16, 1999

DERWENT-ACC-NO: 1999-254219
DERWENT-WEEK: 199930

TITLE: New MDM2-specific antisense oligonucleotides
INVENTOR: AGRAWAL, S; CHEN, J ; ZHANG, R

PRIORITY-DATA:
1998US-0073567
May 6, 1998
1997US-0916384
August 22, 1997

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 9890237 A	March 16, 1999	N/A	000	C12N015/11
WO 9910486 A2	March 4, 1999	E	057	C12N015/11

INT-CL (IPC): A61 K 31/47; A61 K 31/70; C07 H 21/00; C12 N 15/11

ABSTRACTED-PUB-NO: WO 9910486A
BASIC-ABSTRACT:

NOVELTY - Antisense oligonucleotides (AN1) that inhibit MDM2 protein expression are new.

DETAILED DESCRIPTION - AN1 binds to MDM2-encoding RNA and is complementary to a sequence that overlaps, by at least 1 nucleotide, a sequence (I) (defined in the specification) within the MDM2 RNA.

INDEPENDENT CLAIMS are included for the following:

(1) an antisense oligonucleotide (AN2) that inhibits MDM2 protein expression, having the sequences represented by (N1) and (N2);

(2) an antisense oligonucleotide (AN3) having one of the 20 or 21 bp sequences fully defined in the specification;

(3) a method for activating a tumor suppresser, particularly p53, in a cell by contacting the cell with an antisense oligonucleotide complementary to a portion of MDM2-encoding RNA; and

(4) a method for synergistically enhancing DNA-damage induced activation of p53 in tumor cells by contacting the cell with a DNA-damage inducing agent and an antisense oligonucleotide complementary to a portion of MDM2-encoding RNA.

5'-GATCACTCCCACCTTCAAGG-3' (N1)

5'-TGACACCTGTTCTCACTCAC-3' (N2)

ACTIVITY - Tumor-suppressing.

Choriocarcinoma JAR cells were grown in DMEM medium supplemented with 1% FBS according to standard cell culture techniques. Cells were then treated for 30 hours with growth medium containing 400 nM of either antisense oligonucleotide (N1) or control oligonucleotide (N3) and 7 mu g/ml lipofectin (Gibco BRL Paisley, UK). Treated cells were then photographed using a phase contrast microscope.

The results showed that the antisense oligonucleotide induced significant cell death. Dying cells showed the morphology characteristic of apoptosis. The control oligonucleotide induced significantly less apoptosis.

5'-GATGACTCACACCATCATGG-3' (N3)

MECHANISM OF ACTION - Inhibition of MDM2 expression.

Choriocarcinoma JAR cells containing wild-type p53 were grown in DMEM medium supplemented with 1% FBS according to standard cell culture techniques. Cells were then treated for 18 hours with growth medium containing 50, 100. 200, and 500 nM of various antisense oligonucleotides including (N1) or with control oligonucleotides (N4) or (N3) and 7 mu g/ml lipofectin (Gibco BRL Paisley, UK). Treated cells were harvested and lysed, and total protein extracted according to standard methods.

2 mg total protein were mixed with 100 mu l of hybridoma supernatant containing an anti-MDM2 monoclonal antibody 2A10, and with 20 mu l of packed protein A-Sepharose beads (Sigma, St. Louis, MO). Immunoprecip itates were obtained by incubation at 4 deg. C for 3-5 hours on a rotor. The beads were then washed with lysis buffer three times. Immunoprecipitates were then boiled in loading dye, and samples were fractionated by electrophoresis on an SDS polyacrylamide gel with a 5% stacking gel and 10% separation gel. The gel was transferred to an Immobilon P membrane (Millipore, Bedford, MA). The membrane was then blocked with PBS/5% non-fat milk+1/500 polyclonal serum for 1 hour. The membrane was then washed with PBS/5% milk and I125 protein A (0.2 mu Ci/ml) for 1.5 hours, then with PBS and 0.1% Tween20 and exposed to a phosphorimaging screen.

Treatment with the antisense oligonucleotide above resulted in approximate ly 3-5 fold inhibition of MDM2 expression at concentrations of 100-400 nM. The effect was not observed with control oligonucleotides.

5'-CAGAGCCTTCATCTTCCCAG-3' (N4)

USE - AN1, AN2, AN3 are used to inhibit tumor growth in a mammal, including a human, particularly in conjunction with a DNA-damaging agent such as camptothecin (claimed).

ADVANTAGE - Unlike prior art phosphodiester oligonucleotides, the invention provides oligonucleotides that are useful as investigative tools and as potential therapeutics.

Term	Documents
1 NEAR3 (3 OR 2)	33

including document number

Display Format: